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EDITORIAL

THE ORIGIN AND SPREAD OF INFECTION

THE infectious nature of some diseases, one may cite leprosy as an example, has been recognised by the layman from time immemorial. It would also appear that the ancient Persian veterinarians had a clear conception of what is implied by the term "infection," but this knowledge was lost during the Middle Ages. Morton (1722) believed that the original cause of consumption "may possibly be some species of animalcula or wonderfully minute living creatures that by their peculiar shape or disagreeable parts are mimicable to our nature." Others made shrewd guesses, but it was the Italian, Bassi (1835), who first clearly demonstrated the parasitic nature of infectious disease. It was not, however, until the latter part of the nineteenth century, following on the work of Pasteur, Koch and their contemporaries, that the true nature of infection began to be understood and generally recognised. At about the same time the transmission of various protozoan diseases by insect vectors was being demonstrated. In this field, Smith and Kilborne's work (1893) in the transmission of Texas fever is classical.

Following on these discoveries the application of hygienic and immunological methods produced enormous strides in the control of epidemic disease, but the hope that a specific organism and a specific vaccine would be found for every disease was doomed to failure. Nevertheless, when the concept of deficiency diseases, due to the lack of minute amounts of essential food substances, was put forward it was received with extreme scepticism.

It is both remarkable and fortunate that most of the great "killing" epidemics of man and animals have been relatively easy to control either by hygienic or immunological methods. We are now afflicted with a number of less spectacular but none the less serious diseases which often cannot be controlled

by the classical methods. The various forms of mastitis in cattle may be taken as an example. Whilst, in the main, the chronic form due to *Str. agalactiæ* may be regarded as an infectious condition spread from udder to udder of lactating cows, it is known that the number of heifers infected at the first calving is much higher than was previously thought. Infection with numerous serological types of *Str. agalactiæ* has been reported during the first lactation period in newly established heifer herds under experimental conditions, and it is known that when fed on milk containing *Str. agalactiæ*, heifer calves may become infected by sucking each other's teats. After examining a large number of naso-pharyngeal swabs from normal human beings, one group of workers concluded that all the common pathogens—pneumococci, hæmolytic streptococci, diphtheria bacilli and others—could probably be isolated at some time or another from every normal person if the swabbings were made carefully and frequently enough. There are indications that the same conclusion would hold true of the organisms causing mastitis: so-called latent infections are recognised to be common in chronic mastitis and it is known that the other mastitis streptococci, staphylococci and *C. pyogenes* can fairly easily be isolated from the tonsils or vagina, and Lovell (1943) discussed the possible endogenous origin of mastitis due to *C. pyogenes*. The common respiratory diseases of man and animals are also difficult to control and the view is gaining ground that they are all primarily due to virus infections, although they are complicated by streptococci and various other organisms which are normally found in the naso-pharynx. The exact origin of these virus infections is uncertain; discussing human influenza, Burnet and Clark (1942) point out that when strangers visit an isolated community there is always an outbreak of respiratory infection and from this conclude that human carriers must be common. They are sceptical of Shope's evidence that, during inter-epidemic periods, the virus of swine influenza survives in the eggs of the common lung worm. It is certainly well recognised that whenever "green" horses are stabled with a group of healthy seasoned horses, particularly if the hygienic conditions are bad, the young animals develop various respiratory affections.

The epidemic disease of animals which attracts most attention in this country is foot and mouth disease. After studying the spread of this infection one is not surprised that there were many who clung to the idea of the spontaneous origin of disease: it must be admitted that in many outbreaks we have no idea how the animals could have become infected. Stockman and Garnett were the first to bring forward evidence that the incidence of foot and mouth disease is related to the movement of migratory birds; Bullough (1942) has collected further evidence. It would appear that the prevalence of foot and mouth disease in Great Britain, which occurs chiefly in the autumn and winter, is closely related in time with the arrival of starlings from continental areas where the disease is common; it is more prevalent and occurs earlier in the south-east. Nearly all the starlings migrating to Scotland came from Norway, where foot and mouth disease is rare, and it is well known that Scotland has for many years been remarkably free from infection. Further, in Sweden the greatest incidence of foot and mouth disease is in the summer, instead of the autumn and winter, but again this corresponds with the arrival of migrating starlings. It is, of course, fatally easy to correlate the incidence of a disease with the occurrence of

some natural phenomenon: the reason for the winter prevalence of influenza in this country is no better understood than that of foot and mouth disease, but it can easily be correlated with various other occurrences, particularly atmospheric. When discussing Stockman and Garnett's evidence that the prevalence of foot and mouth disease was due to the movement of migrating birds, Thomson (1924) said that unknown factors in the pathology of the disease might equally well contain the clue to an explanation of quite another kind. We do not know, for instance, why foot and mouth disease is non-existent in Finland. Further, Eccles (1939) failed to demonstrate virus on starlings obtained from infected premises, and had great difficulty in setting up the disease by flying large numbers of birds from infected to healthy animals. One feels, however, that the virus must behave differently under natural than under experimental conditions.

Of recent years there has been some change in the incidence of foot and mouth disease; outbreaks in the summer months have been more common and the number of outbreaks in Scotland has increased. Considerable publicity has been given to the view that infection is due to the feeding of unboiled swill, but Rutherford (1942) claims that this cannot be the whole explanation, because out of 60 "initial" outbreaks during the last three years only three have been in pigs and five in pigs-and-cattle. It is clear that there is still much to be learnt about the origin and spread of disease and the methods of control.

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General Articles

TALK ABOUT MILK

By LIONEL J. PICTON, O.B.E., M.A., B.M., B.Ch.
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(SCENE: *The Resident Surgical Officer's Room in a great Hospital. The R.S.O. is discovered reading in front of the fire. Two visitors are announced—a portly elderly man, and a Naval Officer.*)

R.S.O.: Oh, hello! I got your card and thought you might make it to-night.

1ST VISITOR (*a Country Doctor*): This is my brother. Though you've known me so long you've never met him——

R.S.O. (*to Naval Officer*): But I've heard of you, Sir; and of your interest in farming. The doctor says that when you're not a sailor you're a farmer.

NAVAL OFFICER: That's about right. My brother says he's come to talk to you about milk; and as I'm most interested, may I sit and listen—just as *amicus curiæ*? I won't interrupt.

COUNTRY DOCTOR: I hope you will; there should be three in any talk. But let me get this off my chest. (*To R.S.O.*): Here's your last letter; you say (*reading*): "Since I've been in this place it has been gradually borne in upon me that the dangers of tuberculous milk, which we used to dismiss so lightly, are to be taken very seriously; and that the people who want to pasteurise all milk may very likely be right."—Well, I love to get your letters; but that bit made me feel sad. As a country doctor, I can't help knowing the facts: you know them too, yet, after all, you come to this "Wen," and it looks as if you have become corrupted.

R.S.O.: Well, you'll soon retrieve me, talking like that of Cobbett and the countryside. But I'm going to resist your rural magic for a bit. This morning we were operating on a volvulus and—it had nothing to do with the volvulus, but—I noticed the iliac glands were like a string of beads, hard, calcified, like yellow ivory; and the surgeon said: "There's raw milk for you."

COUNTRY DOCTOR: So he implied that they were caused by drinking raw milk?

R.S.O.: Yes. And that this bovine tuberculosis is a scourge that would be prevented by pasteurising.

COUNTRY DOCTOR: How did he know that this was bovine?

R.S.O.: Come to think of it, he didn't; but they pretty well all are, aren't they? Isn't almost all surgical tuberculosis bovine?

COUNTRY DOCTOR: This R.S.O.'s room of your's is pretty well stocked with books——

R.S.O.: Old standards mostly——

COUNTRY DOCTOR: Yes; but good and to the point sometimes. (*Pulling out two red volumes.*) Here's John Fraser's "Surgery of Childhood." He gives the late Dr. Stanley Griffiths' figures——

R.S.O.: My word, you are quick on the draw!

COUNTRY DOCTOR: Take glands in the neck, first in children under 10: fourteen of the cases were due to human type bacilli, against 33 due to bovine.

R.S.O.: Well, that's in favour of our honorary's view.

COUNTRY DOCTOR: Wait a bit: in little people over 10, no less than 41 were human against 21 bovine. Now bones and joints; in the under-tens 217 were human type and only 71 bovine; while in the over-tens 172 were human against 16 bovine.

NAVAL OFFICER: Those are impressive figures. I'm bound to say, as a lay man, the impression I have got from common talk is that people think tubercle of all sorts is spread by milk and that sanatoria for consumptives could be almost closed down if all milk were pasteurised. I've supposed that that was an exaggerated view; but that there was something in it.

COUNTRY DOCTOR: It's well that I brought my pistol to this party. (*He produces a letter.*) Here's a letter from Dr. Peter Edwards, of the Cheshire Joint Sanatorium. I asked him this very point, that is, the percentage of cases of lung infection due to bovine tubercle, because I knew he had the answer, for he and his friends, Dr. Lyn and Mr. Cutbill, have typed no less than 2,300 cases. It seems the results have never been published: but what a solid authoritative mass of figures it is! The paper is coming to birth shortly; but he's sent me a typescript, reporting on the first 1,315, which will do for our point. Of these, 1,282 were human, one was "dysgonic human" (you must translate that, I'm off the irons with this bacteriology) and 32 were bovine. That's less than 2½ per cent. bovine: all the rest were human.

R.S.O.: Well, that confirms the idea that pulmonary tuberculosis is mainly due to human bacillus.

COUNTRY DOCTOR: Yes, but there is more to it than that. Listen to this: "It is the rural population which provides the greater number of bovine infections."

NAVAL OFFICER: Isn't that rather against your argument? I suppose the country people drink more milk and so get more often infected.

COUNTRY DOCTOR: No, that's not the inference. Look at the figures: in an urban population of a million and a quarter there were 13 bovine lung infections, whilst in a rural population of about 16,000 there were 19.

NAVAL OFFICER: Sorry, but those figures seem to favour my argument.

R.S.O.: Read on through—there were 13 + 19—thirty-two bovine infections in all. (*Reading over the shoulder of C.D.*): "Of the 32 bovine cases only six were habitual milk drinkers.

COUNTRY DOCTOR: Yes, Dr. Edwards told me several of them wouldn't even have milk in their tea. Well, then, where did they pick up the germ? See this table: it is suggestive.

Amongst these 1,315 patients 2.43 per cent. had the bovine strain. Fifty-nine of these were cattle contacts (farmers, butchers or tanners), and of these 19 per cent. had the bovine strain, whilst of the 1,256 non-cattle contacts only 1.7 per cent. had the bovine strain. It's contact "as does it"!—not milk in these cases.

R.S.O.: Here's another pointer. They had eight farmer patients, and five had the bovine strain. It is evident that the breath of the infected animal is

the source of the farmer's infection. Well, that does alter the perspective a bit.

COUNTRY DOCTOR: Let's step back and have a look around the problem. There's a bit in Edwards' letter, which he sent with the typescript, which I think pretty good—here it is: "This little article more or less justifies my statements over 25 years; namely, that there is more harm done by the lack of milk rather than by dirty milk. People have assumed that milk is the cause of a lot of bovine infection; and, secondly, on the small figures published they have assumed that a large number of people die from bovine infection, whereas this has not been proved. For example, if I had published at the end of 300 cases of typing, I would have said there were over 7 per cent. in this area" (bovine pulmonary, he means), "whereas at 500 I was down to 2.2 per cent., and ever since the figures have rarely varied between 2 per cent. and 2.5 per cent." Then he goes on about an individual patient who had the human type bacillus: "After many months, to our amazement, we were able to culture a bovine germ as well. . . . I would not be surprised if I publish it, that those who want pasteurisation would say it was due to the fact that we use raw milk. So we do, and use it from the cleanest byres in the countryside. Actually we have never been able to get more than about 5,000 organisms in a c.c. of this milk. Furthermore, the cattle are vetted regularly and followed to the slaughter-house. Never has there been any sign clinically or post-mortem of tuberculosis in this herd."

NAVAL OFFICER: There's a lot in the papers about "safe" milk. Dr. Edwards' milk sounds "safe" enough: but what does the term mean?

COUNTRY DOCTOR: Milk which does not give rise to bacterial disease in humans or calves. A pretty negative definition!

R.S.O.: Well, yes; but why do you stress that?

COUNTRY DOCTOR: What ran through my mind was that calves fed on pasteurised milk have been known to become tuberculous. I think it likely that raw milk from healthy cattle well fed on the produce of the farm—and that's a point—has a protective power.

NAVAL OFFICER: So "protective milk" would be better than "safe milk"?

COUNTRY DOCTOR: Yes, that's my idea, but only as a working hypothesis. To establish that is a great task for the future.

NAVAL OFFICER: It seems to me we've got to ask about this "safe" milk, what bacteria might make it unsafe; and how do they get in?

COUNTRY DOCTOR: Well: (i) Tubercle bacilli, streptococci and Brucella Abortus, from the udder. (ii) Streptococci, diphtheria bacilli or typhoid group bacilli from the mouth or hands of the cowman or dairymaid, or indeed anyone handling the milk. (iii) Typhoid from the water used in the dairy.

NAVAL OFFICER: Then since disease-producing organisms can reach the milk at any stage in its journey from cow to consumer there is no single step, such as pasteurising the milk, which will make it "safe"?

COUNTRY DOCTOR: That's so. Indeed, by heating the milk to 145 deg. Fahr. for 30 minutes, as is done in the holder pasteurising method, all the harmless lactic acid bacilli are killed as well.

NAVAL OFFICER: Wasn't there some story, 50 years ago, that people in Bulgaria

lived to a great age through taking sour milk?

C.D. and R.S.O. (*simultaneously*): Metchnikoff!

COUNTRY DOCTOR: Yes, that's it, Metchnikoff thought these sour milk germs antagonised the miscellaneous and often pathogenic germs in the bowel. Of course, there is truth in the idea; but, apart from what happens to swallowed milk, the lactic acid germs in fresh milk, though they eventually cause souring, antagonise any casual germs that get into the milk.

R.S.O.: So that, if after the milk is pasteurised, fresh organisms get in, such as thermophyllic bacteria from the pasteurising plant, or streptococci from the dairyman with tonsillitis, or typhoid bacilli from the fingers of a typhoid carrier, these organisms have a virgin soil in which to grow, unopposed as they would be in fresh milk by the harmless lactic acid bacilli.

NAVAL OFFICER: "Thermo-phyllic bacteria": that will mean, I suppose, some sort of germs that love heat. Are there some like that and do they do any harm?

COUNTRY DOCTOR: Yes, indeed, there are, and they've been the scourge of some pasteurising plants, difficult to eradicate. The harm they cause is gastro-enteritis.

R.S.O.: So pasteurising, by lending an appearance of safety to milk so treated, deceives the public.

COUNTRY DOCTOR: Yes, it is likely to hold up public demand for healthy herds. I heard from my opposite neighbour last night a quaint bit in his little girl's letter from school. Vera had read in the papers of this proposal to pasteurise—as you know, she is "farmer" through and through—"I should think a man who pasteurises good milk is a fool, whilst one who pasteurises bad milk is a rogue."

NAVAL OFFICER: Good for Vera!

R.S.O. "Out of the mouth of babes . . ."! I quite see what the result of pasteurising would be, a sense of false security would prevent adequate steps being taken to rid herds of tuberculous cattle. For *that* public funds would be needed, and it seems cheaper to heat the milk than kill the cow. But *could* herds be ridded of tuberculosis?

COUNTRY DOCTOR: Undoubtedly, and as our herds are, potentially, the best in the world, it is well worth doing. We have a variety of means, proved successful elsewhere. To do the job in the same way here would need funds and organisation, and intelligent propaganda to explain the issue to the public, who must pay, and to the M.P.s, who must vote the money. Had the energy that has been spent on advocating an out-of-date makeshift, like pasteurisation, been used to eliminate tuberculosis from the herds, the job would have been done by now. The eradication of tuberculosis is a worth-while issue, whilst pasteurisation is a dead issue and leaves us with tuberculous cattle and a tuberculous soil, and milk containing dead tubercle bacilli and sometimes live ones.

R.S.O.: Then why was pasteurisation introduced?

COUNTRY DOCTOR: To provide big milk distributors with a milk which would "keep" for a few days, so that the lactic acid bacilli should not sour it

XUM

during distribution. As an afterthought, the medical officers of health backed it as a safety measure.

NAVAL OFFICER: Does pasteurisation harm milk?

COUNTRY DOCTOR: Yes. It's especially detrimental for infants, and the young, for it reduces the vitamin content, and the lactalbumin and available calcium. Here! I put it crisply in an old health report; I was once that extinct monster, a part-time M.O.H. (*Taking out of his case a slim volume.*) "Not only does heating milk spoil it as a scurvy preventer, it also spoils it from performing its important function as a provider of lime. For instance, in one experiment at the Rowett Institution at Aberdeen, a group of calves receiving pasteurised milk actually contracted rickets." And we've already hinted that it harms milk by making it the perfect medium for growth of accidentally added organisms which flourish in the absence of lactic acid bacilli, but do not always reveal their presence by obvious souring.

R.S.O.: Then what do you advise, Sir?

COUNTRY DOCTOR: Examination, thorough and repeated, of every herd by veterinary surgeons. Such a clinical examination would result in the removal of all dangerously infected cattle.

NAVAL OFFICER: I suppose those would be cows with tuberculous udders?

COUNTRY DOCTOR: Those are far the most dangerous, and of course to sell the milk of a cow so diseased is illegal. But all dangerously infected cattle should be removed. Even if the udder is all right, a few live germs may come over into the milk. And then look at Dr. Edwards' finding: a cow with tuberculosis of the lung may give it to her owner through her breath.

But pasteurisation can't stop that! And pasteurisation can't be confidently relied on to eliminate the tubercle from the milk. Nor does it kill the spores of the germs that cause digestive disturbances in children.

R.S.O.: Then your programme is veterinary inspection—"Attested Milk," isn't it called when the vets certify it?

COUNTRY DOCTOR: That's an important part of my programme; but there's a new vole bacillus vaccine which I think should be thoroughly tested. There's another technical safeguard which I do not think deserves the reliance placed in it, the double intra-dermal test. It seems it is no more than a test for sensitivity to tubercle protein. P.M.s have shown that it is not evidence of active infection.

NAVAL OFFICER: What about the handling of the milk?

R.S.M.: I think it should not only be cooled, but kept cool; organisms will not multiply between 32 deg. and 38 deg. Fahr., and chilling is the ideal method of keeping milk bacteriologically clean whilst it is distributed. It causes no alteration in the nutrient qualities—(trout eggs have gone to Australia in ice and hatched out)—but neither extraneous organisms nor those native to milk will multiply in "chilled" milk.

NAVAL OFFICER: All that sounds very good; but now and then, when, at distant intervals and distant places, I've come in contact with milk production, the same thought has come to me, that heavy milk yields and record-breaking production damage the cow's stamina. And that if optimum, as opposed to maximum, milk production be aimed at, more home-grown feed should be

produced and used—indeed, in these war conditions it is being produced—and that this would and will result in an increase of stamina, (a) because it's more nutritious, and (b) because the artificial stimulus to production is withdrawn.

COUNTRY DOCTOR: That's true and very important; and there's another side to it. When I read Sir Albert Howard's little article in the *Journal of Agriculture*, some years ago, on "Indore compost," I said to myself, here's the key of the whole matter. Then I got his book, and when the Oxford Press printed his "Agricultural Testament" I felt that the way is open to grow crops so healthy that they will not only have disease resistance themselves, but will confer it on the animals that eat them. Mr. Sykes, of Andover, now says that, by this means, in one generation, disease can be abolished from a farm. One might have looked forward to gradual improvement over a number of years, but in one generation, it is startling. I've not seen the farm; but, though I believe it is supremely good, it's one in a line of many which have proved the same principle. Captain Moubray—they call him the "Compost King" in Southern Rhodesia—reduced the death rate of his calves from 30-40 per cent. to 3-4 per cent.

R.S.O.: I've never heard you talk before of Mr. Sykes and Captain Moubray, but I remember you saying the principle would involve giving as much attention to the decay of organic matter and its return to the soil as has been given in the past to the growth of the crops that we get out of the soil.

NAVAL OFFICER (*half to himself*): It's no good fighting nature, as it were; you must fight with it. . . .

(*The porter knocks at the door with an urgent summons for the R.S.O. and the others leave.*)

THE OVARY: AND HORMONE THERAPY

By HAMILTON KIRK, M.R.C.V.S.

IN the realm of endocrinology, a very great deal of work and most of the literature has been devoted in the last year or two to the female sex hormones, and our knowledge of the various ovarian and synthetic œstrogenic substances has been amplified and clarified.

The ovaries produce two distinct hormones—one from the follicle and one from the corpus luteum. There are several modifications of each generic hormone, all having slightly different chemical formulæ. Those from the ovarian residue and follicular fluid are known as the "œstrogens." The luteal hormones will be described later.

The existence of an œstrogenic substance, generically termed "œstrin," was first proved in 1923 by Doisy,[1] who used an alcoholic extract of ovary. It proved impossible to obtain the active substance in pure form, and therefore nothing more was done in the matter until Ascheim and Zondek, in 1927, showed that there was a considerable amount of œstrogenic substance in the urine of

pregnant women. It was later found that there were still larger quantities in the urine of pregnant mares, and from this source a pure crystalline principle was obtained, in 1929, both in America and Germany. This crystalline sex hormone was called "œstrone" (or keto-hydroxy œstrin).

Shortly after, Prof. Marrian—now of Edinburgh University—was able to extract from pregnancy urine another form of œstrone, which was termed "œstriol" (or tri-hydroxy œstrin).

Subsequently it was found that œstrone could be hydrogenated, resulting in the product known as "œstradiol" (or di-hydrofolliculin).

Since œstradiol can be extracted direct from sows' ovaries, it is considered by some that this may be the form in which it exists in the ovary, in which case œstrone and œstriol would have to be regarded as degredation products.

Both œstrone and œstradiol can be converted into esters, and the one principally used is the benzoate. Thus both œstrone and œstradiol benzoate are used for injection, particularly where a large dose is required, because they are absorbed more slowly than the non-benzoated hormone.

Whilst œstrone and œstriol are similar in composition, and practically identical in pharmacological properties, the second-named differs from œstrone on account of its stability in the gastro-intestinal tract. This property of œstriol, coupled with its greater solubility in water, ensures its prompt absorption and therapeutic action after oral administration. The effect of œstriol on œstrus is apparently more prolonged than that of œstrone.

Functions of the Ovary

The ovaries, pituitary, thyroid, and perhaps other endocrine glands, together with the sex centres in the brain, all influence the maturity of the female and her ability to reproduce.

At the onset of puberty and œstrus, several follicles begin to ripen. These ripening follicles produce a "follicular" hormone which stimulates the development of the secondary sex organs of the female, such as the uterus, vagina, mammæ, and initiates a periodic endometrial proliferation of the uterus.

Reaching maturity, the Graäfian follicle usually ruptures, and ovulation occurs. The granulosa cells of the follicle, together with the inner thecal layer which surrounds it, then unite to form the corpus hæmorrhagicum, from which is derived the corpus luteum.

When ovarian function proceeds in orderly fashion a luteinising principle (progestin) is produced by the corpus luteum. The main purpose of progestin seems to be to stimulate the secretory function of the endometrium and to prepare it for the reception of the ovum.

The follicular and luteal hormones are antagonistic in that, whilst the former controls ovulation, the corpus luteum inhibits it between the œstrual periods and during pregnancy. On the other hand, both of them appear to be directly concerned with the regular appearance of œstrus.

The luteal body persists for periods which vary with each species of animal (about five months in the bitch) and then gradually disappears. Following such disappearance œstrus returns again and so the cycle continues.

But if, during œstrus, conception takes place, the corpus luteum becomes

established for a much shorter period (some weeks in the bitch), meanwhile supplying the body with an internal secretion which seems to be indispensable to the maintenance of a correct hormone balance.

If, in the non-pregnant bitch or other female animal, the corpus luteum persists, then œstrus fails to recur at its normal and regular time. This has been proved by veterinary surgeons over and over again, for by expressing the corpora lutea (in cows) they have been able to re-establish the normal sequence of the œstrus cycle.

We can thus reasonably infer that the luteal hormone is antagonistic to œstrus and that it may be the principal cause of suppressed œstrus in many cases met with in practice.

Suppressed œstrus in bitches is a disorder concerning which the practitioner is very frequently consulted, and if an examination of the animal fails to reveal any obvious cause for this abnormality, it is rational to implicate the ovary and proceed upon the lines indicated above, i.e., the administration of those endocrines which are antagonistic to the luteal hormone and which are calculated to stimulate and expedite the œstrual period, viz., the œstrogens.

Discussing Lipschutz's work upon "The Internal Secretion of the Sex Glands," Smith[2] made some very interesting observations. He said: "A chapter is devoted to the experimental proof of the sex-specific action of the testicular and ovarian hormones. If these are specific for each sex, and maleness depends upon the testicle and femaleness upon the ovary, it ought to be possible experimentally to reverse the sexes; and this *has* been done! It is possible to feminise a castrated male guinea-pig by an implantation of ovary, and to masculinise a castrated female by an implantation of testicle. It is almost romantic to read that the rudimentary male teats and mammary glands may not only hypertrophy, but secrete milk and actually suckle the young. Nor is it less astounding to learn that a castrated female guinea-pig, on which a successful testicular graft was made, developed a hypo-spadic penis and prepuce from its clitoris and sheath, though the organ did not attain the size and shape of the true penis.

"The psycho-sexual behaviour of this masculinised female is purely male; it follows females in heat, and fights an ordinary male rival."

The Actions of Œstrin

The ovaries, through their hormone, have an undoubted influence upon other endocrinous organs, a statement for which there is ample proof. They antagonise, for instance, the parathyroids, and should the ovaries be hyperactive they tend to decrease the calcium content of the blood and induce osteomalacia. The latter condition, at all events, is considerably benefited by removal of the ovaries.

As was pointed out when discussing the thymus, disappearance of this gland permits the functional development of the ovary, whilst its persistence is responsible for sex underdevelopment.

The pituitary, adrenal and thyroid glands appear to be synergistically related to the ovary, and are directly opposed by another group consisting of the thymus, corpus luteum, placenta and mammary glands.

Whilst the ovary is at rest and œstrus is in abeyance, such as occurs in

XUM

pregnancy, then the placenta and mammæ come into being. Similarly, the corpus luteum is at this time in the zenith of its importance. Ovarian activity, with its attendant œstrus and ovulation, cannot be resuscitated until the mammary function declines.

The ovary has an important bearing upon the development of secondary sex characters; upon the regulation of growth and development, and upon the control of metabolism. The whole ovary presides over the phenomenon of œstrus and probably determines the length of pregnancy.

One of the effects of œstrin is to stimulate growth of both the teat and the mammary gland. Another is to promote growth of the vaginal epithelium, and in this respect it is known that genital and breast carcinoma can be produced in rodents by means of œstrogens.

There is, however, no justification for apprehending that such a calamity could occur from its normal therapeutic use in the bitch or cat.

Therapeutic Indications

An extract of the whole gland is definitely indicated in hypo-ovarism following oöphorectomy. Removal of the ovaries is often a cause of mammary atrophy and shrinkage of the uterus and external genitals. It favours a tendency to obesity with some subjective nervous symptoms. The glands are not essential to life, however, and their removal may be effected with impunity.

In the human being, it is said that oöphorectomised women lose the feminine voice, grow hair on the upper lip and chin, and acquire a straighter and more masculine contour. Nevertheless, the writer knows at least three such women who have, some years after the operation, still shown none of these signs.

An interesting series of figures by Richter from studies of the metabolism in a castrated bitch showed that the metabolic activities, which had been considerably reduced by the operation, were remarkably augmented by the administration of a preparation of ovary.

This seems justification for the results reported by Sicard and others, to the effect that obesity frequently is due to hypo-oöphorism and that it often may be treated advantageously in this manner.

Anœstrus and feeble œstrus are conditions against which the œstrogens are particularly powerful; similarly, delayed or irregular œstrus may usually be rectified by the administration of either the follicular hormone or an extract of whole gland.

In general, the indications for the use of ovarian preparations are those in

Whole Gland.	Œstrin.	Progestin.
Anœstrus.	Anœstrus.	Anœstrus.
Frigidity.	Anaphrodisia (female).	Abortion (threatened).
Hysteria.	Oligomenorrhœa.	Menorrhagia.
Labour (inducement of).	Sterility (functional).	Metrorrhagia.
Obesity (female genital).	Pruritus vulvæ.	Sterility (functional).
Oöphorectomy.	Delayed œstrus.	Painful œstrus.
Sexual underdevelopment.	Irregular œstrus.	Deficient milk secretion.
		Hyperthyroidism.

An anonymous F.R.C.V.S. writes: "I attended a bitch which suffered from constant *uterine hæmorrhage* and was followed continuously by male dogs, yet which was unable to take part in coitus. After eight doses only of ovary and thyroid, the hæmorrhage cleared up (chronic condition as it was) and all organs previously affected became normally dry. The normal blood flow was re-established and the bitch became pregnant."

which the animal's symptoms are suggestive either of derangement of the gland or deficiency of its secretion.

Whether luteal extract, follicular or whole gland should be employed, must be decided more or less upon our knowledge of their various functions.

Veterinarians may usefully try the various extracts as indicated in the list of disorders at foot of previous page.

It has been observed that "there are extra-genital causes of *sterility* which can be corrected by internal secretions, notably that of the thyroid. The avitaminoses, and especially iodine insufficiency, play a role in the genesis of ovarian sterility. Implantation of ovaries may have two opposite effects: it may activate or it may inhibit follicular development in the ovary of the animal grafted. That is, if the grafted ovary contains a fresh corpus luteum it will render the grafted animal sterile for a time; but if it contains no corpus luteum, the inactive ovaries of the grafted animal become active.

The lesson to be learned from these observations is that where ovarian implantation cannot be carried out the sterile animal should be given ovarian hormone in oil subcutaneously, and preferably combined with thyroid.

Boyd, an American veterinarian, observed that the practitioner who is experienced in the treatment of sterility fully appreciates the value of the dislodgement of the corpus luteum in the successful treatment of pyometra. He stated that:

> "In cows, where the disease has been present a short time, the corpus luteum will frequently be found near the surface of the ovary, but in the long-standing cases the yellow body will be found to be more centrally located, and therefore more difficult of ablation. The dislodgment of the corpus luteum in its entirety is responsible for such rapid changes in the uterus that one wonders at so much power being invested in so small a structure. Not only does the uterus promptly discharge its contents, through the re-establishment of muscle tone, but it also changes rapidly, morphologically, regaining its symmetry and normal anatomical positions within a very short time."

Here again, the deduction is that where ablation of the corpus luteum is not possible, the administration of hormones which are antagonistic to that of the corpus luteum, is the next best procedure.

Zupp and Murphey,[3] in their article on "The Phenomena of Œstrus," stated: "We have been doing some experimental transplantations, but doubt the necessity of this difficult procedure except in senile cases where the œstrual hormone has failed, since cases of functional *sterility* may often be cured by a single injection of œstrual hormone. . . . In post-mortems made upon animals in which the sterility was believed to be of endocrine origin . . . we have found diseased conditions of both the hypophysis cerebri and thyroid glands."

Synthetic Œstrogens

Similar to the natural œstrogens, the synthetic œstrogen known as "stilbœstrol" produces almost all, if not all, of the effects of the natural ones. Lazaro[4] found that, except in rare instances, stilbœstrol does not produce any demonstrable enlargement of the uterine cavity, but it has definite œstrogenic clinical effects. Mazer[5] and others stated that "the effectiveness of stilbœstrol, *given orally*, is in marked contrast to the inactivity of the natural œstrogens when so administered which, in this way, lose 95 per cent. of their potency.

Although some disagreeable side-effects may have been observed in humans,

XUM

it does not necessarily follow that they would appear also in animals similarly treated. The writer has so far received no such reports, nor observed them personally. Mazer found that stilbœstrol given hypodermically was successful in six out of seven cases of amenorrhœa in women. The oral administration of 5 mg. daily for ten days was successful in all of seventeen attempts.

Anterior pituitary-like œstrogenic hormones have been synthetically produced from pregnancy mare's urine, and are in great demand at the present time.

Another gonad-stimulating hormone (Serogan) has been prepared from the serum of pregnant mares. It has functions similar to those of natural œstrin; and it is claimed that in the male it stimulates the germinal epithelium of the testes, leading to marked increase in spermatogenesis.

Administration and Dosage

Ovarian products may be employed as a soluble extract or as a solution in oil for subcutaneous or intramuscular injection. This applies to whole ovary, ovarian residue (ovary from which the luteal body has been extracted) and to corpus luteum. The synthetic stilbœstrol is generally given per os, whilst stilbœstrol dipropionate is injected. Following are the chief ovarian products and their dosages:

Œstrin, or follicular hormone, typical proprietary representatives of which are "Unden" and "Œstroform." Unden is a solution in oil, 1 c.c. of which is biologically standardised to contain 200 International Units of hormone. The oral dose is 10 m. daily to a medium-sized dog, for about ten to twelve days following the commencement of a feeble œstrus. By the expiry of that time the bitch should accept the dog.

Another representative product is an aqueous solution of "Theelin," 1 c.c. of which contains 200 I.U. This is injected subcutaneously or intramuscularly in doses of approximately 1 c.c. daily, for the same purposes.

Œstriol (represented by Theelol) in capsules containing 0.06 mg. of crystalline hormone, for oral administration. Each capsule equals 200 I.U. and is the equivalent to 1,000 I.U. of Theelin *given orally.* Dose: One capsule every three days.

Œstrone (Theelin in oil). 1 c.c. of the oily solution equals 1,000 I.U. for subcutaneous or intramuscular injection. It is very slowly absorbed and therefore has a sustained and continuous effect. Dose: 1 c.c. bi-weekly.

Œstradiol Benzoate (Œstroform in oil) for oral administration. Each c.c. equals 1,000 International Benzoate Units. This product is also injected, and is even more slowly absorbed than the non-benzoated hormone. Dose: 1 c.c. weekly.

Desiccated gland in tablet form, and vaginal suppositories, are also available, each (Theelin) suppository containing 2,000 I.U. The dose of desiccated whole gland for a dog is 5 to 15 grs. once or twice daily.

In order that ovarian administration should affect a future "heat," it should be commenced three or four weeks before œstrus is expected, and in all cases it should be persisted with for a considerable time.

There appears to be a well-founded belief among those progressive practitioners who have been trying out the œstrogenic hormones that ovary in combination with thyroid has a more definite effect than when given alone. In fact, there is a general disposition to add anterior-pituitary to the prescription, all three glands being so profoundly synergistic.

Michell,[6] Salmon[7] and others have stated that the implantation of œstrogens in the form of loose crystals or compressed tablets was more efficacious than the injection of similar amounts of hormone in oil. The method was found quite satisfactory and safe for the treatment of primary anœstrus with lack of mammary development. Implantation affords a long-continued administration which is not so perfectly or economically obtained by the introduction of watery solutions or suspensions at intervals. It would also counter the obesity following upon bilateral ovariectomy.

The Corpus Luteum

As already explained, the yellow-body known as the corpus luteum is the end product of the ripening of the Graafian follicle at the time of œstrum. It persists in the bitch for about five months, then gradually disappearing.

Only in the absence of its influence can œstrus again recur. The hormone of this body, therefore, is antagonistic to that of the ovary.

Its endocrine is known variously as Progestin, Progesterone and Corporin.

Progestin is used as a general term to indicate the hormone of the corpus luteum without reference to the state of chemical purity; whilst the term *Progesterone* is used to designate the chemically pure substance having the structure and properties described later.

The work of Fraenkel, who demonstrated that the removal of the corpus luteum from animals in early pregnancy was followed by absorption or premature expulsion of the fœtus, forms the basis of the modern pharmacology of progestin.

From the time the ovum is fertilised until the birth of the offspring, the complementary and also the antagonistic actions of the œstrogenic principle and progestin are at work.

Progestin prepares the endometrium for the nidation and nourishment of the fertilised ovum, subsequent to the preliminary influence of the œstrogenic principle.

Progestin is necessary for the conservation of early pregnancy; it maintains the uterus in a state of quiescence during pregnancy, and the uterus does not become sensitive to the oxytocic principle of the posterior lobe of the pituitary gland until the influence of the corpus luteum declines.

As gestation advances, the amount of œstrogenic substance increases, reaching a maximum at term. According to this theory it is in this way that the balance between the œstrus-producing principle and progestin is disturbed; the œstrogenic hormone becomes dominant and sensitises the uterus to the oxytocic action of the pituitary, and labour ensues.

The most characteristic activity of progestin is seen in its effect on the endometrium. When this structure is previously stimulated to hyperplasia by the œstrogenic hormones, progestin induces a secondary type of growth and develop-

XUM

ment which are characteristic of the early days of pregnancy and of the pre-gravid or pre-œstrual state.

Another effect of progestin is also believed to favour gestation. It inhibits uterine contraction, thus safeguarding the position of the fertilised ovum or fœtus. In this respect the effect of progestin is antagonistic to that of œstrone, which sensitises the uterus to the stimulating action of Pitocin. Whilst progestin inhibits uterine contractions, œstrin increases them.

The administration of progestin counterbalances the œstrogen dominance which exists in cases of excessive bleeding due to primary ovarian dysfunction. Rapid cessation of bleeding has been reported to follow the administration of from one-half to 10 I.U. of progestin daily. Functional menorrhagia may, in fact, be an example of progestin deficiency.

Progestin, together with the œstrogenic hormones (e.g., extracts of whole gland) induces growth and development of the mammary gland, and an increase in milk secretion.

Because the corpus luteum inhibits ovulation, it is a probable (or possible) cause of sterility.

When habitual abortion is not accounted for by other factors, and appears to be caused by endocrine deficiency, progestin may often be utilised to advantage. Administration should be commenced at least a week before abortion is anticipated, as based on previous history.

Progestin in vegetable oil (Lipo-Lutin) is administered preferably by intramuscular injection into the buttock. It cannot be given orally as it produces no effect by this route. One c.c. of Lipo-Lutin equals 1 International Unit (I.U.); or a preparation of double strength may be obtained if desired. Dose: 1 c.c. bi-weekly.

Progesterone has been produced synthetically. *Pregnandiol* is a substance chemically related to it and exists in the urine of pregnant mares. It is actually derived from progesterone and represents the latter's excretion product. In the normal œstrual cycle pregnandiol appears in the urine a day or two after ovulation.

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TRICHOMONIASIS IN THE COW—II

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(*Concluded from page* 106)

Experiments in Artificial Infection

THE experimental work recorded here is the clinical side of a much more extensive research done in collaboration with Dr. M. Robertson.

EXPERIMENT C

Clinical Aspect

Apart from tentative experiments with individual animals this constituted the first controlled experiment of a group of heifers exposed to infection with *Trichomonas fœtus*.

Nine virgin heifers were bought and maintained for several months before the actual experiment commenced in August, 1939. This was done to enable the collection of sufficient data relating to their normal blood picture, as judged by a series of agglutination tests for *Trichomonas fœtus*. On several occasions these animals were tested against *Brucella abortus* with negative results.

The Constitution of Experiment C

Group A.—Four heifers, C1, C3, C7 and C8, mated by the "clean" bull C9. An intravaginal injection of 10 c.c.s of a washed suspension of *Trichomonas fœtus* strain Belfast was made immediately after service.

Group B.—One heifer, C4. A similar method of infection was followed as in Group A, except that the trichomonad strain used was "Utrecht."

Group C.—Two heifers, C2 and C10, mated by the bull C9, which had been artificially infected some five days previously with a washed suspension of *Trichomonas fœtus* strain Belfast.

Group D.—Two heifers, C5 and C6—controls.

Group A

C1.—Mated by clean bull C9 and immediately after service was injected intravaginally with *Trichomonas fœtus* strain Belfast. From the third day a vaginitis developed which reached its peak on the ninth day. It was acute and characterised by hæmorrhagic papules around the clitoris and lines of inflammation on the lateral walls of the vagina. These pencil lines of inflammation were distinct and in marked relief to the more normal vaginal tissue. From the ninth day this acute inflammation subsided and was replaced by a dark reddish diffuse vaginitis with a well-marked proliferation of the surface lymphatics covering the lateral walls of the vagina. This gave them a harsh feel on palpation. The discharge at first was brown in colour, later changing to golden yellow, and profuse, but later diminishing in quantity. After the third day trichomonads were not seen until the sixty-seventh day, although constant search was maintained. On the sixty-seventh day rectal examination of the uterus showed the left horn to be larger than the right and quite soft on palpation. Gentle massage

of the uterus per rectum expressed approximately 100 c.c.s of a greyish-white fluid rich in trichomonads (7½ millions per c.c.). This was followed by slight changes in the mammary gland and in the genitals suggestive of an early abortion. This phase passed off and the heifer settled down to continue normally the remainder of her pregnancy.

The vagina returned to normal shortly before her calving time and remained normal thereafter.

C2.—This heifer had first been injected intravenously, the amount of antigen given containing 15 million trichomonads as a first dose and increased during seven weeks to 500 millions. These amounts were shown by later experiments to be low to immunise so large an animal.

Before immunisation this heifer had a titre level equal to that found in normal sera. The titre rose to well above normal, the index of the highest sample being 17/8. (The ratio is here made with the serum of the animal itself before the start of immunisation.) The highest point showed a titre amounting to that considered a true positive. It was, however, evanescent and dropped quickly to a degree only somewhat above normal. Thereafter it remained at this level.

This heifer was served by C9, which might have been infected at the time of service, but was never shown to be so. The evidence is against this bull having ever acquired an active infection. C2 had a healthy calf at full time.

C3.—This heifer was mated by clean bull C9. She was infected into the vagina immediately after service.

At seven days the vaginitis, acute in nature, made its appearance preceded by the hæmorrhagic points around the clitoris. At ten days it became more acute and the lines of inflammation had become more sharply defined. This vaginitis remained to the twenty-first day, when œstrus returned. During the whole of this period of twenty-one days trichomonads were present. A greyish-white discharge persisted throughout, becoming more profuse on the approach of œstrum. After an interval of six weeks C3 was mated and successfully conceived and at nine months calved normally. The vaginitis throughout pregnancy remained chronic until shortly before calving time, when the vaginal mucous membrane returned to normal.

Trichomonads were not seen subsequent to the second mating.

C4.—The preparation of this heifer was the same as C1 and C3, except that the trichomonad strain used was Utrecht.

The symptoms that developed were similar to those in C1 and C3. At the seventh day the vaginitis was acute and followed a similar course, to terminate in a chronic granular vaginitis.

At six and a half months the vaginitis again became acute and some twenty-eight days later and fifty-eight days before normal calving was due *Trichomonas fœtus* appeared in the vaginal discharge in large numbers. This discharge, milk-like in consistency and comparatively profuse, lasted seven days. The trichomonads decreased in numbers after the first day and from the seventh day onwards were not seen again. The uterus, palpated per rectum, appeared normal.

The heifer completed her pregnancy and calved normally a healthy calf.

Examination of the discharges following parturition failed to show the presence of trichomonads.

C5.—This was a normal control, mated by C9 and calved normally. She was examined regularly but showed no vaginitis and no trichomonads.

C6.—This heifer acted as a media control, sterile broth being injected into the vagina immediately after service.

C6 followed a course similar to C5 and calved normally.

C7.—This heifer was in the same group as C1, C3 and C8, and was injected with *Trichomonas fœtus* strain Belfast.

A less acute course than in the case of C1 was experienced. No trichomonads were noted after the second day following infection, until the fourth month, when, for three days, a few relatively inactive and degenerated forms were observed. From this time until she calved normally the parasite was not seen again, nor was it found in the discharges following the birth of the calf. The scanty number of trichomonads seen in the fourth month is the only positive microscopic finding, but is unmistakable evidence that this heifer harboured trichomonads for four months.

C8.—This heifer was treated as C1, C3 and C7.

No reaction took place until the fifth day, when a mucoid pus was present and contained numerous trichomonads. The vaginitis which appeared on the fifth day was very acute, the pencil lines of inflammation being well marked as well as the groups of petechial hæmorrhage around the clitoris. The discharge in this case was golden coloured. During the following seven days the discharge changed to the more customary greyish-white mucoid type and the trichomonads became fewer and more inactive until at the twelfth day they disappeared. The vaginitis had also changed in nature to the more chronic and granular form as found in the other heifers in this group.

At six weeks œstrum returned, no trichomonads being then noted. Four months later active trichomonads appeared for two days associated with a slight increase in the vaginal discharge. The chronic vaginitis remained throughout the period of pregnancy. At nine months a weakly calf was born. The discharges following parturition were profuse, brown in colour, and unhealthy in appearance. However, no trichomonads were seen in these discharges.

Thus, trichomonads were harboured for certain for the first four months of pregnancy.

C9.—As a clean bull he was used to mate all the heifers with the exception of C2 and C10. Following these matings 10 c.c.s of a washed culture of trichomonads, strain Belfast, was injected into his sheath. At five and seven days later C2 and C10 were mated.

In view of the experience gained by the author in examining known infected bulls, it would appear that the injection of 10 c.c.s quantity of washed culture had failed to infect this bull. The experiment, though cited, is therefore of a negative character.

C10.—This heifer was mated by bull C9 seven days after he had received an injection into his sheath of *Trichomonas fœtus.*

This heifer pursued a normal pregnancy and finally calved without showing evidence of trichomonads or of any form of vaginitis.

This heifer was later included in the A experiment, where a positive infection was produced (*vide* A experiment).

Summary of Experiment C

The results may be summarised as follows:

Five heifers were infected into the vagina with *Trichomonas fœtus* immediately after service with clean bull C9. All the animals were successfully infected and harboured the parasite for varying times.

Four of the heifers (C1, C4, C7 and C8) were rendered pregnant by this first service and all produced live calves at full term.

One heifer, C3, came into season after three weeks but on being mated two months later became pregnant and calved to time.

The trichomonads were harboured as shown by microscopical findings for various periods.

Heifer C4, infected with strain Utrecht, showed trichomonads only for seven days at 6½ months, but must have retained that infection since the period of service.

C3, which returned into season at three weeks only, showed the parasite during the first sixteen days after being inoculated. It should be noted that two œstrus cycles intervened before she was finally mated and during this time the heifer may have got rid of the trichomonads.

C7 retained the trichomonads for at least four months, but only showed them in sufficient numbers for observation.

C8 harboured the parasites for five months but they were seen only at rare intervals.

This infection, therefore, did not result in the sterility of the animals, nor did it cause any of them to abort. It is, in fact, an example of a latent or mild degree of infection producing vaginitis and some disturbances but not rising in any of the animals to a continuous acute infection.

The injection of trichomonads into the sheath of the bull did not result in a recognisable in any of the two heifers served after the injection, namely, C2 and C10. There is, in fact, no evidence that an infection was established.

EXPERIMENT A

Clinical Aspect

In this experiment the animals were divided into four groups, as follows:

Group I.—Two heifers, A1 and A7—controls.

Group II.—Four heifers, A4, A5, A6 and C10, mated by the clean bull A9. An intravaginal injection was made immediately after service of 10 c.c.s of a concentrated washed suspension of *Trichomonas fœtus* strain Belfast.

Group III.—Four heifers, A2, A3, C4 and A11, mated by the artificially infected bull A9.

Group IV.—One heifer, A8, previously immunised before being mated by the infected bull A9.

It is not proposed to discuss in this section details of the experiment or of

its serological results other than to give a brief clinical report of the infected animals. The full details will be published in a further paper.

Group I

The two heifers, A1 and A7, were mated by the clean bull. They pursued an uneventful pregnancy period and calved normally. The vaginal secretions were examined at frequent intervals with negative results.

Group II

A4.—The trichomonads were demonstrated in the vaginal secretions for the three days following the artificial infection. They were not seen again during the pregnancy period nor in the abomasum of the dead calf or the uterine discharges. In seven days after service the characteristic vaginitis developed associated with lines of acute inflammation and hæmorrhagic nodes. The vaginitis later developed into the chronic granular form which was a dominant feature of these experiments.

A5.—Trichomonads were not seen after the first twenty-four hours until the twenty-first day, when they were numerous and remained so until œstrum returned at the forty-first day. The vaginitis at this stage was very acute. The profuse discharge was rich in epithelial cells and active trichomonads. The second mating was carried out by the now positively infected bull A9. Following this mating the vaginitis still remained acute but trichomonads were not observed, œstrum returning 18 days later. A third mating was made by the bull, which at this time was almost incapable of service. A5 conceived to this mating and calved a live calf at the normal time.

Trichomonads were only seen in the period between the first and second mating, although the vaginitis persisted for a considerable time.

A6.—The trichomonads were not seen after the second day. The vaginitis was comparatively mild and the vaginal secretion never profuse. This animal calved a healthy calf at the normal time.

C10.—In the C experiment this animal was mated by the artificially infected bull but failed to show evidence of infection. 200 c.c.s of a 1/400 iodine solution was injected into the uterus of C10 before inclusion in the A experiment.

No vaginal disturbances were noted subsequent to the artificial infection until the eighth day, when a moderate vaginitis with a small amount of discharge was evident. At the ninth day trichomonads made their first appearance and were observed daily until the twenty-first day, when œstrum returned. C10 was mated and remated sixteen days later. During this second period of sixteen days trichomonads were not observed. Vaginitis was definite and the discharge profuse. Trichomonads were present for two days only, forty days after mating. At this stage the discharge was profuse, flaky and purulent. Œstrum returned fourteen days later and C10 was again mated by the now positively infected bull. Œstrum returned in thirty-eight days. During this period the vaginitis and the vaginal discharge were less acute and the trichomonads made sporadic appearances only. At this stage C10 was treated with iodine.

In this experiment C10 developed a characteristic endometritis and returned to service five times. *Trichomonas fœtus* made definite but sporadic appearances throughout the course of the experiment.

XUM

Group III

A2.—Following service, the vaginal disturbances were moderate and trichomonads were not seen until forty-four days later, when they were both numerous and active. Œstrum returned at the forty-eighth day. Eighteen days after the second service trichomonads reappeared in large numbers. The vaginitis was acute and the discharge profuse. Two months later œstrum returned. This animal was rendered temporarily sterile. Rectal examination failed to show evidence of changes in the uterus.

A3.—Trichomonads were not seen after the first day. The heifer calved a healthy calf at the normal time. The vaginal disturbances were mild and the vaginal secretion negligible.

C4.—In the C experiment this animal showed numerous trichomonads at the seventh month of her pregnancy and thereafter calved normally. Subsequently C4 was treated by injecting 200 c.c.s of a 1/200 solution of iodine into the uterus.

This animal was mated by the infected bull A9. Seven days later an acute vaginitis developed, accompanied by a small quantity of discharge. Thirty-three days later active and numerous trichomonads appeared in the discharge, which at this stage was profuse, fluid and whitish. Œstrum returned at five weeks. Following the second mating, trichomonads were not demonstrated again, although the vaginitis and profuse discharge remained. Œstrum returned approximately two months after the second service. No further matings were made, as the bull was incapable of further service.

A11.—Some degree of vaginitis followed the mating by the infected bull, but trichomonads were never demonstrated in the vaginal secretions. This animal calved a healthy calf at the normal time.

Group IV

A8.—This heifer had been vaccinated by a series of intramuscular injections with trichomonas. She was exposed to infection by injecting a washed suspension of *Trichomonas fœtus* into the vagina. A8 did not develop a trichomonas infection and a normal calf was born at full time, although some degree of vaginitis was present.

In the A experiment many types of the clinical cases of trichomonas as seen in the field were produced. Again it was demonstrated that, as in the C experiment, the trichomonad made definite but sporadic appearances in the vaginal secretions of infected animals.

Summary

The symptoms and lesions of trichomoniasis as found in Northern Ireland are described. The outbreaks in the main are characterised by breeding irregularities and endometritis, and may follow a mild or latent course with occasional exacerbations manifested by a greatly increased degree of sterility. Pyometra is not a feature of these outbreaks.

Consideration is also given to the very important aspect of its diagnosis. Experience has shown in Northern Ireland that the agglutination test as a herd

test in association with the herd history is sufficient evidence to reach a diagnosis of trichomonad infection.

Various aspects of the iodine treatment are discussed as the result of 15 years' experience in the use of this operation.

Included also are the clinical results of artificial infection in fifteen heifers with *Trichomonas fœtus*. These results were characterised by examples of many of the types of clinical cases as seen in the province. A predominant feature was the definite but sporadic appearances of the trichomonads in the vaginal secretions.

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VETERINARY JURISPRUDENCE

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VETERINARY surgeons are familiar with their duties under Statutory Law, i.e., the law derived from legislation, such as the Disease of Animals Acts, etc., but with their responsibilities under Common Law they may not be so well acquainted.

Common Law is used in two senses:

(1) The law derived from custom as distinct from that derived from legislation.

(2) The law administered by Courts of Common Law as distinct from that administered in Courts of Equity, i.e., Chancery Courts.

Actions coming under Common Law are in *Tort*, or on *Contract*. What are these?

(1) *Torts* are wrongs which have nothing to do with contracts, but which infringe legal rights. They may be said to be injuries arising from an infraction of duty. They are injuries or wrongs suffered by one person at the hands of another, irrespective of any contract between them, but distinguished from a crime as not being a wrong done to the community as well as to an individual, and hence not punishable under the Criminal Law.

(2) *Contracts* are agreements enforceable at law.

Negligence. This is a tort, and it has been defined as: "Being under a duty to show care, and doing that which a reasonable man would not do, or not doing that which a reasonable man would do, and thereby causing damage."

Professional negligence includes more than is found in this definition, for, in addition to the duty to show care, one must show competence and skill. In undertaking the surgical and medical treatment of animals for remuneration, a veterinary surgeon is offering service to the client, and, as such, he owes a duty to take care and apply such skill as he possesses.

It is the breach of this duty that constitutes professional negligence, and in the case of a veterinary surgeon it must indicate the absence of that measure of care and skill which may reasonably be expected from anyone in his position.

How much skill and care does the Law require to be exercised?

Measure of Skill. When he puts himself before the public in his professional capacity there is raised the implication that a veterinary surgeon is warranting his possession of competence and the necessary skill. He is thus proclaiming to the public that he possesses what the Law requires in skill and ability, and it follows, that, if he is found to lack this amount of skill, i.e., a fair, reasonable and competent measure of it, he will be rendering himself liable. It would be left to the jury to determine this lack of skill, and decide whether what is complained of resulted from it.

Measure of Care. This must be of a character and amount to deserve the description of being reasonable in the light of all the circumstances prevailing. The Law insists on reasonable care, and a veterinary surgeon would be prudent always to observe this to an extent that at any time he could go before a Court and testify that he bestowed as much care and application as any other veterinary surgeon would have done if he had been placed in a similar situation.

To avoid liability, a veterinary surgeon should be careful to get his client's consent to any serious operation; he should not undertake the treatment of a case if the circumstances are unfavourable without warning the client; he should give his grooms, dressers, etc., all necessary warnings and instructions regarding any case; and he should avoid introducing incompetent aid into his practice, because he is liable for any lack of skill in his assistant.

To be on the safe side a practitioner should have his client's consent to his sending an assistant or locum tenens, because by so doing he will not be liable for their negligence if he has taken reasonable care in selecting qualified persons competent for the work. A practitioner leaving a student in sole charge of his practice is running a risk—both he and the student could be liable. If a principal employs an unqualified assistant or locum tenens he runs a grave risk. To avoid any allegation of improper treatment it is safest to prescribe what is usually given and approved of in the profession. For any experiment, or the application of a new treatment, the veterinary surgeon can guard himself by getting the approval of his client; otherwise, if things go wrong, he may be in danger of having damages given against him in any action for negligence. This should not be looked upon as a bar to being progressive, for this has been laid down in a Court:

"The Law demands qualification in the profession practised, not extraordinary skill such as belongs to few men of rare genius and endowment, but that degree which ordinarily characterises the profession; but the client is entitled to the benefit of the increased knowledge of the day. The standard of ordinary skill is on the advance; and he who would not be found wanting must apply himself

with all diligence to the most accredited sources of knowledge."

When a charge of negligence comes before a judge and jury, the judge will decide if the duty existed, and the jury will decide how much care is usual and reasonable.

All the foregoing will sound, to the busy practitioner, something of a rather terrifying nature. However, he may reassure himself, for of all the difficult people to conduct a successful action against, the professional man is not least.

In any allegation of negligence, to make a veterinary surgeon liable, a client will have to prove against him that he showed a want of competent and ordinary care and skill, and to such a degree as to have led to a bad result. A simple error of judgment is one thing, and the lack of care and skill another. The rule being, if the veterinary surgeon exercises care and skill, he will not be liable for a simple mistake in judgment.

(*To be continued.*)

Review

Topographical Anatomy of the Dog. By O. CHARNOCK BRADLEY, M.D., D.Sc., F.R.C.V.S. Revised by TOM GRAHAME, T.D., F.R.C.V.S., Professor of Anatomy Royal (Dick) Veterinary College, Edinburgh; Lecturer on Comparative Anatomy, University of Edinburgh. Fourth edition. Oliver & Boyd, Ltd. Edinburgh: Tweeddale Court. London: 98, Great Russell Street, W.C. Price 28s.

BRADLEY'S "Topographical Anatomy of the Dog" has earned its place amongst the classics of veterinary literature. When it was first published its object was to fulfil a long-felt want amongst graduates and undergraduates, whose attention was being directed more and more to the maintenance and care of the dog. As with all efforts to break new ground, it did not satisfy all, and there were many critics. Subsequent editions, however, saw great improvements and this, the fourth edition, has in no way lagged behind. It is fortunate that the publishers have been able to retain the services of Prof. Grahame, in order to revise the work. He was for so long associated with the author that one could be sure that the Bradley touch would not be lost.

In this he has succeeded, and, further, by rewriting certain parts and introducing new material he has enhanced its value to the reader.

Many new illustrations have been added and old friends redrawn and clarified. Amongst the former one would draw attention to those showing muscular attachments, the brachial and lumbo-sacral plexus, and the series of X-ray photographs. The latter are particularly welcome, for veterinary surgeons are often called upon to read X-ray films, and it is necessary to have a sound knowledge of the normal before one attempts to give an opinion on the abnormal.

A special section has been devoted to the lymph nodes and system, a method of approach which has much to commend.

As in previous editions, the book is not divided up into sections or chapters.

XUM

It is a continuous story from start to finish. However, an excellent index is available for anyone who may desire to make inquiry on any particular point or feature. Prof. Grahame has done his work well and we can with every confidence say: "This is a book that no student or graduate can afford to do without."

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